CASE REPORT

Coma as a presenting symptom of primary HIV infection

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Primary HIV infection (PHI) is symptomatic in 50–90% of patients. The diagnosis, however, is seldom made at first presentation. This is probably because of the multifaceted and unspecific manifestations, the omission to perform adequate diagnostic testing and the failure to assess risks for PHI. Meningoencephalitis has been described as a fairly common presenting condition in PHI, with nuchal rigidity, fatigue, photophobia and headache; therefore, PHI should be considered in the differential diagnosis of aseptic meningitis. We present the case of a man with acute coma and a presumptive diagnosis of viral encephalitis in whom serological testing showed HIV encephalitis during PHI.

A 30-year-old male patient presented in September 2005 with a history of diarrhoea, chills, fever with peaks at 40°C and cough with purulent sputum that had persisted for 11 days. He reported having had sexual intercourse with a man, a chance acquaintance, 6 weeks earlier. An HIV test performed 4 weeks before the present admission had been negative.

On physical examination, some pain and stiffness of the neck and a faint macular rash were observed. The patient was agitated and within a few hours his mental state deteriorated to coma, with extensor responses only to painful stimuli.

White cell count was normal, but the differential diagnosis showed reduced numbers of lymphocytes. Flow-cytometric analysis showed 44/µl CD4 lymphocytes. Routine blood biochemistry tests were abnormal for alanine aminotransferase (157 U/l), aspartate aminotransferase (105 U/l) and lactate dehydrogenase (437 U/l). C reactive protein concentration was mildly increased at 17 mg/l.

A spinal tap was performed, which showed pleocytosis of 633 lymphocytes/µl. The results of serological investigations are shown in table 1.

Treponemal tests were negative, as well as tests for toxoplasma, gonococcus, *Mycobacterium tuberculosis*, cryptosporidiae, microsporidiae and bacterial cultures from blood and cerebrospinal fluid (CSF).

Antiretroviral treatment was started with abacavir, lamivudine and zidovudine. The patient's mental state improved rapidly. Within 48 h he was able to follow conversations, was afebrile and the rash had subsided. The patient was discharged 5 days later, and after 4 weeks HIV by polymerase chain reaction was below the detection limit and CD4 cell count was normal.

Table 1 Serological investigations

CMV IgG	Serum	Positive 1:6400
CMV IgM	Serum	Positive
CMV DNA PCR	Serum	Negative
CMV DNA PCR	CSF	Negative
EBV IgG	Serum	Positive
EBV IgM	Serum	Negative
HSV IgG	Serum	Positive 1:19 000
HSV IgM	Serum	Negative
HIV ELISA	Serum	Reactive
HIV I and II Western Blot	Serum	Negative
HIV RNA PCR	CSF	4.84×10^6 copies/ml
HIV RNA PCR	Serum	0.55×10^6 copies/ml

CMV, cytomegalovirus; CSF, cerebrospinal fluid; EBV, Epstein–Barr virus; HSV, herpes simplex virus; Ig, immunoglobulin; PCR, polymerase chain reaction.

DISCUSSION

Two earlier reports describe coma in HIV primary infection. In the first, however, there was no absolute evidence of simultaneous HIV seroconversion,[1] whereas the second described a case of fatal brain necrosis coinciding with the development of a CD8 lymphocytosis, suggesting that the cytotoxic lymphocyte response rather than the HIV infection itself was the cause of neurological disease.[2]

PHI, although symptomatic in most patients, often remains undiagnosed. The availability of routine nucleic acid amplification assays allows the detection of viraemia at an early stage of infection, but the diagnosis may be missed by routine testing, as antibody-based tests for HIV remain negative for several weeks into the course of the disease.[3 4]

HIV displays a well-known tropism for neural tissues. Apart from opportunist infections of the central nervous system and HIV-associated malignant diseases, a variety of manifestations due to the direct action of HIV have been described.[5] These may involve peripheral nervous tissue and the spinal cord, but leucoencephalopathy has also been described. Leucoencephalopathy usually causes a syndrome of cognitive dysfunction known as "AIDS dementia complex". An AIDS-defining disease, it is present in about 10–30% of patients.[6] More specific involvement of brain structures causing distinct focal symptoms can be found.[7]

The signs and symptoms of aseptic meningoencephalitis occur in about a quarter of people with HIV primary infections presenting for medical consultation.[8] Acute confusional states suggesting more severe encephalopathy have been described.[1 9] In our case, the assumption that the neurological symptoms were caused by the direct effects of HIV is based on the fact that no other cause could be found, and that the viral load in the CSF was comparable to the findings published on patients with AIDS and HIV encephalitis.[10] Unfortunately, we could not perform repeated tests of HIV RNA on the CSF of this patient. Neurological

Key messages

- Neurological manifestations are common in PHI.
- Coma can rarely be a presenting sign of PHI.
- The diagnosis relies on nucleic acid amplification of HIV RNA.
- HIV infection should be considered in cases of unclear coma, even if there is no history of AIDS.

Abbreviations: CSF, cerebrospinal fluid; PHI, primary HIV infection

impairment, as well as the rash, improved rapidly after starting antiretroviral treatment. This is suggestive of either spontaneous resolution or a threshold of viraemia to induce coma, but we are unable to provide further evidence for this.

PHI should be considered in the differential diagnosis of coma, and nucleic acid amplification of HIV RNA should be performed, if indicated.

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